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A Case of So-called Laryngeal
Vertigo.

BY
I. ADLER, M. D.,
VISITING PHYSICIAN TO THE GERMAN HOSPITAL,
NEW YORK.

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A CASE OF
SO-CALLED LARYNGEAL VERTIGO.

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THAT peculiar, though somewhat varying, group of symptoms which, following the lead of Charcot, is commonly designated as laryngeal vertigo, is still a subject of discussion as regards both its pathology and its ætiology. The well-authenticated cases thus far reported are comparatively few in number. Scarcely more than two dozen cases have been reported since Charcot first called attention to the affection in 1879, and the authors differ widely in their interpretation of the clinical facts. Under these circumstances every new case of so rare an affection becomes valuable as possibly shedding some light on a point hitherto doubtful or obscure. From this point of view I venture to present the following case:

Mr. J. E. H., aged about fifty-three, merchant. No hereditary taint of any kind. With the exception of several attacks of gonorrhœa, claims never to have had venereal disease. Minute examination detects no evidence of syphilitic lesion. Does not use tobacco in any form. Is accustomed to take several glasses of wine or beer with his meals. He is a man of florid

complexion and robust and healthy appearance. He professes never to have had any serious illness, but is subject to "colds and coughs." From time to time during the last few years he has shown slight but sufficiently well marked symptoms of a gouty tendency and has had occasional attacks of muscular rheumatism. About three years ago he had an attack of acute bronchial catarrh with little or no febrile symptoms, accompanied for several days by thin, foamy, and not very copious bloody expectoration. No pulmonary lesion could be detected, and the sputum then and since remained free from tubercle bacilli. The slight hæmorrhage was referred to congestion of the bronchial mucosa. In November of 1890, in the course of a slight bronchial catarrh attended by rather violent paroxysms of cough, he claims during one of these coughing spells to have suddenly "fainted." Being hurriedly called in and arriving a few minutes after the attack, I found the patient apparently perfectly well and bright. Questions addressed to him and to the members of his family who had witnessed the attack elicited the following facts: The paroxysm of cough had been no worse than usual. He was in a sitting position when the cough seized him, and during the coughing he arose as if to expectorate, then suddenly fell to the floor, totally unconscious. He claims to have noticed no premonition of any kind. There was no giddiness; he was not conscious of any tickling or burning or other sensation about the larynx. The bystanders were unable to state whether the face was pale or turgid or livid. There was no cry, no involuntary micturition, nor were any convulsive twitchings noticed. The unconsciousness lasted but a very few seconds. The patient raised himself without assistance from the floor, laughed at the alarm expressed by his family, felt no lassitude, sleepiness, or any discomfort whatsoever. Careful examination showed some dry râles in the larger bronchial tubes, slight hyperæmia of the pharynx and larynx, and an elongated uvula, otherwise no lesion of any kind. The bronchial catarrh, accompanied by still rather violent paroxysms of cough, passed away in about a week, but no further seizure like the one described occurred. The treatment consisted in the administration of opiates and expectorants.

I desire to note here a peculiarity in Mr. H.'s manner of coughing which struck me whenever I had occasion to see him for one of his periodical attacks of coughing. It seems as if he had acquired a habit of "choking" over his cough whenever the paroxysm is even moderately severe. The chin is depressed so as almost to touch the sternum, head and shoulders stoop forward, the face becomes turgid and red, the superficial veins swell out, and the cough consists of a deep inspiration, succeeded by a number of short, spasmodic expiratory movements following each other in rapid succession, and differing only from pertussis in lacking the long sibilant inspiration which commonly ends the paroxysm in the latter.

During April of this year Mr. H. had an attack of influenza, and following this, after the febrile stage had passed, a very violent cough. At first there were all the symptoms of diffuse bronchial catarrh, sonorous râles over the entire chest, mucopurulent expectoration, etc. Later on the râles gradually vanished, the expectoration diminished, and finally at the end of about ten days ceased altogether, but the cough remained more violent than ever, and assumed a more spasmodic character. The peculiar habit referred to above was still more marked. The cough came in paroxysms, during both day and night, seriously interfering with his sleep. It commenced with tickling in the region of the throat and larynx, became at once very violent, accompanied by turgidity of head and neck, lasting from some seconds to several minutes, and ended usually with the expectoration of a small lump of glassy mucus, sometimes, however, without any expectoration. This state of things continued for about a week, sinapisms, opiates, expectorants, and inhalations being administered without any apparent effect on the paroxysms of coughing. One evening during a violent coughing spell, which occurred while Mr. H. was standing upright, he suddenly dropped to the floor utterly unconscious. I was called in at once and arrived a few minutes after the seizure. There had been no premonitory signs. The cough commenced with the usual tickling in the upper air-passages, but did not apparently differ from any of the preceding paroxysms. There had been no giddiness. The patient did not feel that anything un-

usual was going to happen. He simply dropped to the floor, almost immediately to arise again, feeling perfectly well, but not aware of any unusual occurrence.

The physical condition now was the following: Very moderate granular pharyngitis, elongated uvula, slight hyperæmia of the larynx, heart and lungs in normal condition, pulse full and regular—between 60 and 80 beats per minute. The pupils of equal size and reacting normally. Ophthalmoscopic examination negative. No appreciable symptoms on the part of the nervous centers. The knee-jerk somewhat subnormal, but still sufficiently well marked. The urine, which was examined frequently and carefully, at no time showed albumin, casts, or sugar. The quantity of phosphates and urates was slightly in excess of the normal. No other abnormality could ever be detected. Opiates had been given before; they were now administered in larger doses. No attack during the next two days; then another one of exactly the same character as before, with merely this difference: that, happening to recline on the sofa, he did not fall to the ground, but simply lost consciousness. As opiates had been of no service, they were now replaced by large doses of the bromides of sodium and ammonium, and a competent laryngologist made daily applications of a spray to the pharynx and larynx. The attacks of sudden loss of consciousness following cough now appeared daily, soon several times during twenty-four hours, by night as well as by day. During one of these attacks about this time Mr. H. slightly bit his tongue. The seizures evidently becoming more frequent and more severe, the local treatment, after about ten days of spraying, was discontinued. By the desire of the patient and his friends, the advice of a very well known neurologist was obtained. On recommendation of the latter, iodide of sodium was added to the bromides, and both gradually increased up to ten grammes *pro die*, also gradually increasing doses of the red iodide of mercury, blisters to the back of the neck and laryngeal region, and large doses of cerium oxalate. This treatment was continued for about three weeks. Under the influence of the bromides the pharynx and larynx became quite anæsthetic, and the patient stupid and somnolent. There were marked

symptoms of iodism. The seizures, however, steadily increased both in violence and frequency. He had now as many as four or five in twenty-four hours. In one of these attacks, which came upon him while descending from one floor to another, he fell down stairs and was badly bruised. In another he dropped while standing with a friend in the street and severely hurt his face.

It is to be noted that, while no seizure ever took place without preceding cough, by no means every violent paroxysm of cough was followed by loss of consciousness. The daily coughing spells were quite numerous—sometimes several dozen in twenty-four hours—but, as far as I have been able to ascertain, he never had more than five seizures attended by loss of consciousness in one day. Nor were always the most violent fits of coughing followed by these peculiar attacks. Not infrequently a very violent paroxysm would pass without further consequences, while a comparatively rather mild cough would send the patient into unconsciousness. It is stated, too, that in several instances, when the state of unconsciousness had been of somewhat longer duration than usual, slight convulsive twitchings of the eyes and arms were noticed just before consciousness returned.

As the patient was evidently getting worse, all this treatment was now stopped; no medicine whatever was administered, and when, after about six days, the local effect of the iodide and bromide had completely disappeared, another very careful general and local examination was made. The result of the general examination proved entirely negative. Locally the same appearances were found as above stated. The larynx was carefully examined with a probe, in order to determine the presence of any hyperæsthetic spot as the origin of the convulsive coughs. No such spot was found. As no tangible point of attack could be made out in the larynx, as medicinal treatment had plainly and completely failed, and bearing in mind the experiences of Gleitsmann, who had cured a similar case by removing adenoid vegetations at the base of the tongue, and of Charcot, who effected a cure by cauterizing a granular pharyngitis, I determined to clip the uvula. This was done at once. The patient went home and had no attack for twenty hours;

then two very slight ones in rapid succession, and none since then. The cough continued for some five or six days, having completely lost its spasmodic character, and then disappeared altogether.

The preceding history has been given at great length because it seems to offer several points of interest. We have here a man without organic lesion and of fairly good health, who, after a moderate attack of bronchitis, becomes subject to spasmodic cough, attended by frequent seizures of complete loss of consciousness. There is no aura preceding the seizure, no cry, no involuntary micturition, no hebetude or confusion of mind on regaining consciousness. Unfortunately, it so chanced that I was never able to observe an attack in person. It is therefore impossible to say what was the behavior of pulse and heart during a seizure. The members of Mr. H.'s family, who witnessed quite a number of these sudden losses of consciousness, were invariably startled and alarmed to such a degree as to render them unfit for the purposes of closer observation. It was impossible even to make out with any degree of certainty whether the patient was red or pale in the face. Altogether the witnesses tend more toward the belief that the face was red and turgid. The patient himself describes his sensations during a violent paroxysm of cough as "choking," as "wishing to cough, and not being able to cough out"—a sensation evidently very similar to that described by the patient of Russell.* It seems reasonable to assume from all this that the spasmodic cough was frequently followed by spasm of the glottis. That for this latter the elongated uvula was principally responsible would seem to follow from the failure of all other treatment and the prompt relief from all symptoms after the clipping. In what manner the uvula produced the spasm can not be positively ascertained. I

* *Birmingham Medical Review*, vol. xvi, August, 1884.

am inclined to assume that it was by direct irritation of the *rima glottidis*. The patient's peculiar attitude while coughing would tend to raise the larynx sufficiently to permit this, while the fact of these spasms occurring frequently at a time when the pharynx and superior portions of the larynx were well under the influence of bromide anæsthesia would seem to preclude any other mode of mechanical irritation. It is not impossible that the attack of influenza which preceded these spasmodic seizures may have induced a more than ordinary irritability of the nervous system, and that this may explain why former attacks of cough were never, with but a single exception, followed by similar complications.

Leaving out of consideration those cases in which similar attacks have occurred in the course of locomotor ataxia, as so-called laryngeal crises, and where well-marked anatomical lesions have been found in the track of the pneumogastric and recurrent,* we find widely differing opinions as to the nature of this singular neurosis. Charcot † is inclined to accept this group of symptoms as a disease *sui generis* and analogous to Ménière's disease, the afferent nerve in this instance being the superior laryngeal. Gray ‡ and Massei § consider these attacks to be essentially epileptic. Others again, such as McBride, || Russell, ^ Knight, ¶ and Gleitsmann, †† explain the loss of consciousness and attendant

* Jean, *Gazette hebdom.*, 1876, No. 27. Féréol, *Gaz. hebdom.*, 1869, No. 7.

† *Le Progrès méd.*, 1879, 17. *Revue des sciences méd.*, x, p. 135.

‡ *Amer. Jour. of Neurol. and Psych.*, November, 1882.

§ *Giornale internaz. delle scienze med.*, Anno vi. Abstracted in *Internat. Ctrbl. f. Laryngologie u. Rhinologie*, 1885, p. 21.

|| *Edinb. Med. Jour.*, March, 1884.

^ *Loc. cit.*

¶ *Transactions of the Amer. Laryng. Assoc.*, 1886, p. 34.

†† *Med. Monatsschr.*, i, p. 510.

symptoms by disturbances of circulation in the brain, basing their views on the well-known experiments of E. F. Weber.*

It is not my intention to enter into a detailed discussion and criticism of these conflicting opinions, nor to give an exhaustive review of the cases thus far recorded. All this has been done most fully and ably by Thermes,† Knight, and others. In the case of Mr. H., the complete loss of consciousness and the absence of all vertigo and even giddiness, as well as of nausea and vomiting, seem to preclude all analogy to Ménière's disease. There seems no necessity of ranging our case under the head of reflex epilepsy. No spot or nerve could be found by the irritation of which it was possible to produce an attack. Nearly all cases of well-authenticated reflex epilepsy present well-marked convulsive seizures with all the classical symptoms preceding and following the convulsions. Interesting in this respect are the cases of Schneider.‡

The oft-quoted case of Sommerbrodt,§ in which a large fibroid of the left vocal cord apparently caused true convulsive attacks of epilepsy, can not be considered here, as the patient had had epileptic attacks fifteen years before which were at that time referred to a cicatrix on the right hand and disappeared after the excision of the scar. Evidently this was a case of well-marked "epileptic disposition."

It seems as if all the symptoms in our case could be satisfactorily accounted for by the experiments and theory

* Ueber ein Verfahren, den Kreislauf des Blutes und die Function des Herzens willkürlich zu unterbrechen. Müller's *Archiv*, 1851, p. 88.

† Deux observations de vertige laryngé dans la coqueluche chez les vieillards. *Jour. de méd. de Paris*, 1887, p. 936.

‡ Einige Fälle von geheilter Reflexepilepsie der Nase. *Berl. klin. Woch.*, 1889, No. 43.

§ Ueber ein grosses Fibroid des Kehlkopfes als Ursache der Epilepsie. *Berl. klin. Woch.*, 1876, p. 563.

of Weber. Forced expiratory movements with a spasmodically closed glottis caused increased intrathoracic pressure. Ultimately not only the heart itself to a certain extent, but principally the *venæ cavæ* are compressed, the flow of blood to the heart is diminished and then ceases, a condition of arterial ischæmia and venous hyperæmia ensues in the brain. The pulse becomes weaker and finally disappears altogether, and the heart's action comes to a standstill unless, before this clinax is reached, the glottis is reopened and normal respiration is resumed. That this mechanism can and does produce complete loss of consciousness with total amnesia, and even convulsive twitchings, within a fraction of a minute is established by Weber's experiments on himself. All the conditions given by Weber were present in the case of our patient—the spasm of the glottis with violent, rapid expiratory movements, the turgid face and neck, etc. Had it been possible to obtain a satisfactory record of the pulse and heart, or even of the pulse alone, during one of the seizures, the question could have been settled beyond peradventure. As it is, the case appears in all essential respects analogous to Thermes's second case, where the pulse and heart during the spell of unconsciousness were found so characteristically in accord with Weber's results.

Being still completely ignorant of the true anatomical lesion underlying epilepsy, and the experiments of Kussmaul and Tenner and others making it at least probable that disturbances of cerebral circulation play an important rôle in the pathology of epilepsy, there can be no objection, if one was so inclined, to call the peculiar seizure of laryngeal vertigo epileptoid—epileptoid attacks, however, resulting not from a hypothetic irritation of a peripheral nerve, but from great and sudden disturbance of cerebral circulation.

Finally, it is perhaps worthy of note that in the present

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case again, as in so many before recorded, there is a history of gout and rheumatism. That in all hitherto recorded cases, with but one exception (the second case of Knight), the patients are males, of whom the large majority had passed their fortieth year when they became subject to these attacks, are facts that must be taken into account in the future study of this affection.



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